Hypo-and Hyperagentic Psychiatric States, Next-Generation Closed-Loop DBS, and question of agency

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Open Peer Commentary on "Staying in the loop: Relational agency and identity in next-generation DBS for psychiatry" by Sara Goering, Eran Klein, Darin Dougherty, and Alik S. Widge

Title: "Hypo- and hyperagentic psychiatric states, next-generation closed loop DBS and the question of agency"

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In their interesting work, Goering and colleagues discuss the implications of next-generation deep brain stimulation (DBS) in psychiatry on agency and identity. Even though their final paragraph nicely illustrates the concept of relational agency and how a relational account can involve the neurodevice itself, we identified three pressing aspects that fall short within the authors' evaluations. These aspects all concern the specificities posed by the psychiatric context: First, we emphasize that in psychiatry, pathological processes per se influence agency and that a careful analysis should include effects of hypo- and hyperagentic states, the neurodevice and their interrelation. As a corrective, we argue that in hyperagentic states, neurodevices could even serve to reduce agency. Second, based on the contextual specificities and the added complexity posed by them, we claim that in psychiatry, changing one's personality is at the core of the therapeutic aim. Third, to date, symptoms of many psychiatric disorders are heterogeneous and lack a clearly identifiable neural correlate. The latter reflects a serious signal detection problem closed-loop devices (CLDs) for psychiatric disorders have to solve.

Regarding our first point, we agree with the authors that it is important to protect individuals' personality against negative impacts of neurodevices, when concerns emerge that the neurodevice could reduce agency. However, it is important to keep in mind that in many neuropsychiatric disorders, agency itself is vitiated by pathological processes. Accordingly, patients suffering from these disorders often display symptoms of alienation or inauthenticity. Clinical neurology classifies movement disorders as being either hypo- or hyperkinetic - thereby alluding to a distinct interrelation between disorders and their influence on subjects' experience of agency. As an extension to the classification of hypo- and hyperkinetic disorders in neurology, a similar classification relevant for psychiatric agency can be made: While some conditions can be classified as hyperagentic (i.e. excessive experience of one's own causation and control over events), others are hypoagentic (i.e. reduced experience of causation and control) (Haggard 2017). As illustrated by the (hypoagentic) depression dimension of loss of control (Disner et al. 2011), a pathological process that negatively influences the sense of agency is intrinsic to the condition being treated. The paralyzing effects reflected in generalized uncontrollability, helplessness and apathy resulting in an inability to cope with the external world reflects the stupendous influence of the underlying psychopathology on agency. The same feeling of immobility is portrayed with great candor and precision in William Styron's book "Darkness Visible: A Memoir of Madness" (1991) in which the author describes his descent into a crippling and almost suicidal depression.

Most research on pathology of agency has probably focused on schizophrenia. Schizophrenia involves disturbances of self-agency and selfhood and patients suffering from delusions often belief that their thoughts and actions are not their own but imposed on them by someone. As we have outlined in previous work (Glannon and Ineichen 2016) hyperagentic states may interfere with normal behavior that ordinarily enables one to perform common tasks as evidenced in OCD and schizophrenia. The pathological need for control (in states of hyperreflectivity in OCD) or controlling influences from external agents or objects (in delusional states of schizophrenia) may induce loss of control and mental paralysis. On the other spectrum, hypoagentic states in disorders such as apathy and depression can impair the capacity to perform action plans due to mental fatigue or loss of control. Given the vivid influence of different pathologies on the experience of controlling own actions, a more nuanced analysis is needed on how pathological forms of agency interrelate with neurodevices that aim at supporting patients to re-experience, among other symptom-reliefs, a sense of "normal" agency. Thus, the concern of the neurodevices' potential in reducing agency is too reductionist and doesn't do justice to the complexity of pathological processes that go along with changes of agentic self-experiences.

There is also a difficulty when considering the combined effects of pathological states, the neurodevice, pharmacological treatment and their interrelation. In fact, the neurodevices' potential in reducing agency is not necessarily in all circumstances worrisome. In hyperagentic states, CLDs could even serve to reduce agency. Finally, considering pharmacological effects is just as important as dopamine has e.g. been associated with prosocial behavior, psychosis-proness and schizotypy (Smillie & Wacker 2014) and more generally extraversion and personality.

Our second point is in line with the first argument. The authors write "if you knew that a DBS device might significantly alter your personality, such that you might not really feel like yourself anymore once you had it, you would presumably be reticent to consent to the surgery" and "You presumably try to avoid events that would alter your identity in ways you do not prefer". But the point is that in psychiatry the mere intention of changing one's "personality" is at the core of the therapeutic aim. In addition, chronification of illness and interacting effects of pathology on the personality of patients imply difficulty when wanting to accurately specify what it means to «feel like yourself». It is even more difficult when identity itself is part of the therapeutic target as for patients suffering from dissociative identity disorders. Finally, Baylis' notion that personality changes are not a problem if the individual requests and endorses it, again doesn't factor in psychopathological processes that impact on patients' competencies "to request" or "to endorse".

Our third point refers to fully acknowledging the potential risks and benefits of next-generation CLDs. Here, one has to emphasize that neural loops are hardly ever "closed". Neither are they closed within the nervous system nor between the different bodily systems (e.g. immune, endocrine). Originating from DBS research, one understanding of oscillator-theory implicates the underlying circuits to be dynamically coupled, reentrant and non-linear (Montgomery & Gale 2008). Abundant data is furthermore available on e.g. neuro-immune interactions. Hence, even though the term "closed" is appealing because it suggests safety through clear demarcation, we must acknowledge that these loops are closed only with respect to their use of feedback signals rather than modulating the brain without any tuning capabilities. Moreover and in contrast to latest neuroscientific advances that include pathway-specific or even single cell dependent modulation through optogenetics, DBS systems currently lack comparable degrees of specificity. Because symptoms of many psychiatric disorders are heterogeneous and lack a clearly identifiable neural correlate, there is currently a serious signal detection problem. In epilepsy, where CL-systems are investigated, the onset of pathological synchronization of neuronal firing represents a quite specific signal. However, we currently have no

comparable understanding to identify the neuronal signature of, e.g., a depressive episode that would trigger the stimulation of the device. Finding such signals is likely to entail substantial risks for patients.

Our critical remarks point to a more fundamental problem: Because neuroscience has just started on investigating the mechanisms that generate the sense of agency, there is a need to enrich metaphysical thoughts by empirical research including both explicit (e.g. action-recognition task) and implicit agency measurements (e.g. intentional binding task). Naturally, factors other than a brain-internal device influence the feeling of agency: priming individuals can increase sense of agency while coercion reduces it (Haggard 2017). Studies have additionally shown that both, retrospective inference and the prediction of outcomes are linked to the brains' generation of the experience of agency (Haggard 2017). Future studies should therefore distinguish between prospective and retrospective agency, integrate implicit and explicit tests whilst controlling for confounding factors. That the brain regularly produces a sense of agency through retrospective inferences that lacks or doesn't use a direct signal about the true origins of actions (Haggard 2017), must also be considered. Should CLDs be used for direct modulation of agency, this again exacerbates the signal detection problem. Finally, because the concrete experience of initiating a voluntary action is not sufficient for sense of agency (Haggard 2017) and the controversy of whether patients have a realistic assessment of their (limited) agentic capacity corroborates the need not to focus on patients' self-reports entirely.

Conclusion

Just as the disruption of agency that can be caused by movement disorders, psychopathological processes affecting the sense of agency have implications for well-being. In the end, sense of agency results from brain activity patterns that are being influenced by pathological processes just as much as by therapeutic approaches. In recognition of the complexity, a challenge for future research is to gather data and to develop and evaluate more integrative perspectives concerning the multiple influences on agency and personality including pathology-device interactions.

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